EDITORIAL

NEONATAL CHRONIC LUNG DISEASE

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Advances in neonatal intensive care have decreased the incidence and morbidity of chronic lung disease. However, chronic lung disease remains an important issue for clinical and public health because this illness is associated with chronic respiratory difficulties, prolonged and recurrent hospitalization, and increased incidence of neurological disabilities, growth restriction, and death.

Chronic lung disease following lung injury is one of the most frustrating illnesses in modern neonatal medicine. Despite surfactant replacement therapy and other improvements in neonatal care, there has not been a dramatic decrease in its incidence.

The issues surrounding therapy of neonatal chronic lung disease are complex. Many different therapeutic modalities have been investigated, and a systematic review of controlled trials in the scientific literature is needed in order to determine which therapies have adequate proof of efficacy.

There are numerous studies on the use of parenteral or/and inhaled corticosteroids in the treatment of chronic lung disease. Longer and shorter corticosteroid regimens have been evaluated. Dexamethasone usage has become widespread due to its immediate short-term efficacy, but data concerning long-term pulmonary effects should be obtained.

Non-pulmonary effects of dexamethasone therapy may be significant and can be divided into two categories. The first are usually transient adverse effects that are not difficult to manage clinically. These include hyperglycemia, hypertension, elevation of the neutrophil count, and protein catabolism with associated poor weight gain.

The second group of adverse effects includes those which are less common, but warrant concern because of their potential to adversely effect the ultimate outcome of the infant, such as intestinal perforation and gastrointestinal hemorrhage, increased risk of severe retinopathy of prematurity, an elevated risk of infection, and the suppression of the hypothalamic-pituitary-adrenal axis. Observations of children treated with systemic glucocorticoids that suggest that the risk of growth suppression is decreased when the frequency of administration is reduced also point to effects on the hypothalamic-pituitary-adrenal axis. Furthermore, supporting evidence comes from studies in adults, which have assessed secretory profiles of cortisol during treatment with exogenous glucocorticoids.

The risk of suppression of the hypothalamic-pituitary-adrenal axis depends on the timing and frequency of dosing, suggesting that systemic adverse effects of exogenous corticoids may reflect an interaction with hormonal circadian rhythms. Regarding growth-suppressive effects, the underlying pathophysiologic mechanisms are probably multifactorial, with the involvement of a combination of altered central endocrine and peripheral end-organ responsiveness to many

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physiologic regulators of the growth process.

The timing, dose, route of administration, and duration of therapy required to achieve maximal response with minimal adverse effects in infants with chronic lung disease remain uncertain.

The two most important ways to prevent chronic lung disease, which have been known for decades, are to delay the premature labor and to use prenatal corticosteroid therapy. Research on other methods of accelerating pulmonary maturity are underway. Until then, neonatologists continue to deal with increasing numbers of infants for whom neonatal chronic lung disease is the price of survival.